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VISUAL EVOKED POTENTIALS IN PRIMARY HYPERTENSION

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Abstract : Functional integrity of sensory pathways in the brain has not been well documented in hypertension. It is suggested that vascular endothelial changes including hyalinisation during hypertension may lead to demyelination in the vulnerable areas of the brain. Since optic nerve is considered to be part of brain hence the present study was done to find out if visual pathways are involved in hypertension. Transient pattern reversal visual evoked potentials (VEPs) from 01, 02 scalp regions were recorded in 23 primary hypertensive patients and compared with 14 normotensive control. Of these, six patients showed delayed P1 latencies beyond 99% tolerance limit i.e. Mean + 3 SD of normal. The remaining 17 had latencies of all positive (P1 - P3) and negative (N1 - N3) waves comparable to those of the control group. Correlation Coefficient worked out, showed significant correlation between systolic BP and P1 latency in the control group only. No other parameter showed any correlation with P1 latency & amplitude in both the groups. These findings show that fluctuations of BP in normotensive subjects have correlation with P1 latency. This correlation ceased to exist in hypertensive patients and abnormality in P1 latency of VEP was detected in 26% cases.

Key words : visual evoked potential; pattern reversal; hypertension; visual pathways

INTRODUCTION

Neuronal dysfunction in hypertension might have multifactorial etiology. White matter lesions in brain have been associated with hypertension (1, 2). It has been suggested that hypertension causes vascular endothelial changes including hyalinisation leading onto demyelination and brain infarction. Such demyelination might lead to dementia through disconnection of subcortical-cortical association pathways (3). Similar demyelinating process might occur in vulnerable areas of the brain and cause conductive problems in sensory tracts. Earlier we have reported delayed conduction in auditory pathways in brain stem in grade III hypertension and significant correlation of rise in blood pressure with the absolute peak latencies of auditory evoked response (4). The effect of hypertension on visual evoked potential has not been reported. Marsh & Smith (5) have recently shown that P1 latency of visual evoked response is delayed in pre-eclamptic women. Since optic nerve is considered to be the part of brain, its subclinical involvement is likely in hypertension. It is in this connection the present study involving visual evoked potential was done to see if there was any change in functional integrity of visual pathways in hypertension.

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METHODS

Study was conducted in 23 primary hypertensive and 14 control subjects. Hypertensives were taken from medical OPD of GTB Hospital and controls were the healthy normotensive volunteers. A proforma was filled up giving detailed history of hypertension and its associated clinical features. Before putting them on any antihypertensive drugs, they were given thorough eye check up including refraction, field of vision and fundoscopy to rule out any eye pathology affecting normal vision and tested for visual evoked potentials (VEPs). The criterion of hypertension was three readings of systolic BP > 140 and diastolic > 90 mm of Hg taken on different days. Causal BP was measured with a mercury sphygmomanometer in the right arm in the seated position after 5 minutes rest. All subjects were tested for transient pattern reversal VEPs employing a conventional Evoked Potential Recorder, Visual acuity was tested with Snellen's Chart and corrected to 20/20. Each subject was seated at a distance of 1 metre from the pattern generator screen in dark air conditioned room and was asked to look at the central spot on screen with one eye, other being patched. The VEPs were picked up from 01 & 02 (10-20 Inter-national System of electrode placement) referenced to A1 and A2 respectively with FPZ as ground. The pattern test stimulus on the TV monitor was white & black checks (15 × 15 mm size) subtending an angle of 32 minutes of an arc reversing sides at the rate of 1 Hz. Band pass filter setting was 0.1-100 Hz with automatic artifact rejection. Each eye was tested separately and two sets of 256 responses were averaged. These were analysed by computer of evoked potential recorder (MEB 5200 Nihon Kohden, Japan). The methodology for recording VEPs and technical details employed were similar to those reported earlier (6,7). The peak latencies of negative (N1-N3) and positive (P1-P3) waves along with the amplitude of P1 were calculated. Student's "t" test was used to compare these values between hypertensive and control groups. VEP abnormalities in primary hypertension were identified particularly latencies beyond 99% tolerance limits (Mean +3 SD of normal), being taken as abnormal. Analysis of variance (ANOVA) was done to determine correlation between BP and VEP in normo and hypertensive subjects.

RESULTS

Control group

There were 14 cases in this group ranging from 30-70 yrs. of age with an average age being 36.14 ± 6.95 yrs. They had mean weight of 63.36 ± 10.58 kg, height 161.64 ± 8.41 cms, BP systolic 117.00 ± 7.47 mm Hg and BP diastolic 76.29 ± 6.60 mm Hg. Peak latencies and amplitude of VEPs are given in Table I.

TABLE I: Showing Mean ± SD values of peak latencies of VEP in control and hypertensive group.

Group	*BP* (SYS) mm Hg	(DIA) mm Hg	*Peak latencies of VEP in msec* N1	P1	N2	P2	N3	P3	*Amplitude μv* P1
Control n = 14	117 ± 7.4	76.2 ± 6.6	69.0 ± 5.4	96.3 ± 7.6	124.8 ± 17.5	168.6 ± 17.2	194.5 ± 18.0	216.5 ± 38.3	4.5 ± 1.9
Hypertension n = 23	142 ± 16.8	93.3 ± 8.0	75.3 ± 17.7	103.3 ± 22.4	138.4 ± 28.4	172.3 ± 30.4	209.8 ± 35.6	241.5 ± 40.8	5.1 ± 3.5
'P' value	<.001	<.001	0.138	0.278	0.057	0.527	0.060	0.156	0.597

Hypertensive group

There were 23 cases of primary hypertensive patients ranging from 30-70 yrs of age with an average age of 45.35 ± 12.21 Yrs. They had average weight 60.30 ± 13.97 kg, Height 157.87 ± 9.92 cms, BP systolic 142.09 ± 16.83 mm Hg, BP diastolic 93.39 ± 8.08 mm Hg and duration of high blood pressure ranged from 1-8 yrs average being 3.14 yrs.

The value of P1 latency and amplitude in these hypertensive patients were 103.30 ± 22.46 msec and 5.14 ± 3.52 μv comparable with the normotensive controls being 96.36 ± 7.60 msec and 4.57 ± 1.93 μv.

Out of 23 hypertensive cases, six showed P1 latency beyond 99% tolerance limit, of these four had unilateral and two had bilateral prolongation (Table II). In each group, different physical parameters and BP were compared with P1 latency and correlation coefficients worked out. Only BP showed significant correlation with P1 latency in normotensive subjects (Fig 2).

The representative VEPs of normotensive and hypertinsive case is shown in Fig. 1.

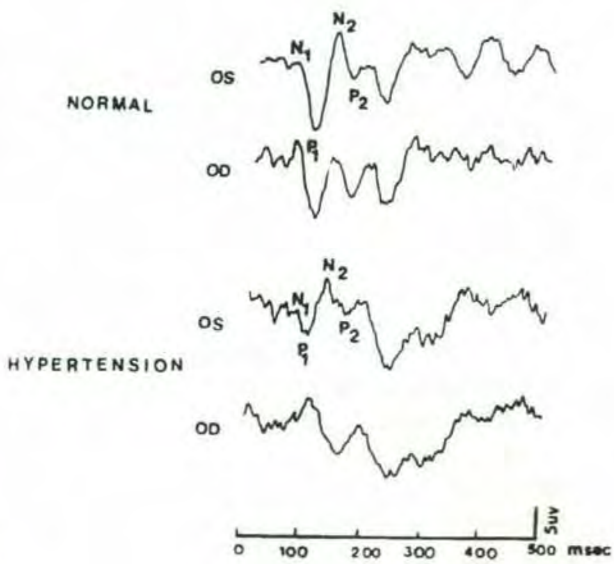

Fig. 1 : Representative tracings of VEPs in normal and hypertensive subject. The tracings of hypertensive subject shows decease in P1 amplitude in left (OS) and delayed latency in right eye (OD)

TABLE II : Showing abnormal P1 letencies beyond 99% tolerance limit in six primary hypertensive patients.

Name	Age (yrs)	Sex M/F	BP (Sys) mm Hg	BP (Dia) mm Hg	Eye	Latency msec	involvement
BKM	59	M	160	100	OS	150	Bilateral
					OD	150	
MSU	41	M	140	90	OS	132	Unilateral
					OD	112	
RSJ	70	M	170	105	OS	142	Bilateral
					OD	152	
SUM	27	F	140	90	OS	136	Unilateral
					OD	100	
YK	36	M	140	95	OS	106	Unilateral
					OD	161	
MKM	60	M	145	90	OS	92	Unilateral
					OD	128	

DISCUSSION

The values of peak latencies of positive P1-P3 and negative N1-N3 waves in our two groups of subjects were similar (Table I). Indeed these values in both control and hypertension group were comparable to already reported values for normal age and sex matched subjects (8).

In our hypertensive group comprising 23 cases, six showed P1 latencies beyond 99% tolerance limit (Mean + 3 SD of normal). This delayed P1 latency was bilateral in two cases and unilateral in four (Table II). The cases having bilateral involvement (BKM & RSJ) belonged to stage II of hypertension as per National Committee criteria of grading (9). This may indicate that higher is the grade of hypertension, more eyes may be involved and involvement becomes bilateral. As we did not have grade III or cases with severe hypertension, this inference cannot be generalised.

These findings suggest that only 6/23 (26%) cases of hypertension had abnormality of VEP. The remaining 17 cases multifocal origin, it is difficult to say which factors have led to involvement of visual pathways in 26% of our hypertensive cases. Sensory derangement particularly of pain threshold have been reported in experimental animals (10) and impairment in sensory conduction in human beings (11) in hypertension. We have also reported delayed latencies of auditory brainstem evoked potentials in the hypertensive subjects (4) and Marsh et al (5) also observed changes in P1 latency in the pre-eclamptic pregnant women. Our finding thus suggest that hypertensive milieu does affect neuronal excitation/conduction in the visual pathways.

It has been postulated that afferent autonomic activity as occurs from carotid baroceptors during normal BP and heart rate fluctuations does modulate sensory inputs. In fact changes in P1 latency has been reported with the carotid pressure fluctuations normally (12). Our observation of negative correlation of BP with P1 latency in the control group (Fig 2) has further supported this finding but this correlation was not seen in hypertensive group. This might be due to resetting of the carotid baroceptor mechanism during hypertension, which resulted in not only loss of this correlation between BP and VEP but also delayed P1 latency in 26% cases.

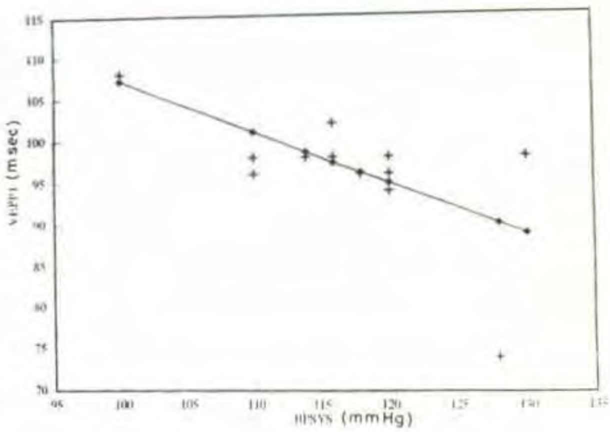

Fig. 2 : Scatter diagram showing correlation between P1 latency and systolic blood pressure in normotensive group. Req. Eq. P1 lat. = − 0.611 BP systolic + 168.125. No such correlation was seen in hypertensive patients.

Further it would be interesting to know if abnormality in VEP obtained in some cases of hypertension is reversible on antihypertensive therapy or not ?

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